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Prevention and treatment of complicated urinary tract infection

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ABSTRACT

A complicated urinary tract infection (UTI) has relapsing and refractory characteristics, and is sometimes life-threatening because of patient predisposing factors as well as the recent worldwide spread of multi-drug resistant bacteria. Patients with complicated UTI should be treated with effective antimicrobial therapy along with appropriate urological intervention to remove predisposing factors when the symptoms are associated. By contrast, routine use of antimicrobial prophylaxis for asymptomatic bacteriuria (ASB) is not recommended, as that would contribute to an increase in even more resistant pathogens. Here, four classifications of complicated UTI, which are considered to be clinically important for general urologists, are reviewed, including UTI in patients with diabetes mellitus (DM) and those with a neurogenic bladder, as well as catheter-associated UTI (CAUTI) and obstructive pyelonephritis secondary to urolithiasis. Appropriate treatment approaches can only be chosen by proper understanding of the etiologies of complicated UTI, as well as correct diagnostic strategies and treatment options.

1. Introduction

A complicated urinary tract infection (UTI), which can involve either the bladder or kidneys, is a symptomatic urinary infection that occurs in individuals with functional or structural abnormalities of the genitourinary tract.[1] Patients with complicated UTI should be treated by effective antimicrobial therapy as well as appropriate urological intervention to remove predisposing factors when the symptoms are associated, such as micturition pain, dysuria, urinary frequency, and low or high fever. By contrast, asymptomatic bacteriuria (ASB) or asymptomatic pyuria is identified by isolation of a specified quantifiable amount of bacteria in an appropriately collected urine specimen obtained from a patient without symptoms or signs referable to urinary infection.[2] Although ASB is usually observed in diabetic women and older individuals, as well as patients with spinal cord injury or an indwelling catheter, routine screening for or treatment of ASB is not recommended.[3]

2. UTI in patients with diabetes mellitus

The prevalence of ASB is three times greater in women with diabetes mellitus (DM) as compared with those without DM.[4] In that study, 26% of nonpregnant female DM patients had significant bacteriuria ($> 10^5$ cfu/mL) as compared with only 6% of the control population. Recently, the significance of ASB in patients with DM was addressed in a placebo-controlled, double-blind randomized trial, which concluded that antimicrobial treatment did not reduce the incidence of cystitis,[5] indicating that the presence of diabetes should not be regarded as an indication for screening or treatment of ASB. However, women with DM are at greater risk of both symptomatic and asymptomatic UTI, although that risk is increased by three–four-fold in females taking oral hypoglycemics or with insulin-controlled DM, indicating a possible association between increased incidence of UTI and severity of DM.[6] Furthermore, UTI in patients with DM is difficult to treat and often recurs, with those patients also reported to have a 40% increased risk of recurrent cystitis.[7] Complications associated with symptomatic UTI are also increased in patients with DM, and women with type 1 DM have increased risk of developing pyelonephritis and subsequent impaired renal function.[8] In addition, neuropathy is a common complication in diabetic patients as the prevalence of neuropathy is estimated to be ~8% in newly diagnosed patients and > 50% in patients with long-standing disease. Genitourinary autonomic neuropathy can cause bladder dysfunction as a voiding problem characterized by poor emptying which could give rise to susceptibility to UTI.[9]

Emphysematous pyelonephritis (EPN) is a severe necrotizing form of acute multifocal bacterial nephritis that results in the presence of gas within the renal parenchyma, with underlying

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poorly controlled DM reported to be present in up to 90% of affected patients.[10] A common clinical trial of predisposing factors is DM, remote or recent kidney infection, and obstruction. It was found that between 85% and 100% of patients with EPN are diabetics, and ~40% have an obstruction most commonly caused by calculi, which is bilateral in 10% of the cases.[11] However, diagnosis of EPN is often delayed because of nonspecific clinical manifestations. Computed tomography (CT) is the preferred method for diagnosis, because of its high sensitivity and specificity for detection of abnormal gas and its anatomical extension.[12] A meta-analysis showed CT was accurate for detecting EPN in all 628 cases, whilst the accuracy rate for plain radiography was 53.2% and for ultrasonography was 67.9%, indicating that, once EPN is suspected, CT must not be delayed to confirm the diagnosis.[13] Treatment of EPN involves broad-spectrum antimicrobial therapy, hyperglycemic control, and adequate urinary drainage with correction of any outlet obstruction. Patients with a necrotizing infection will require a more aggressive treatment approach that includes surgery.[10,14] Once a diagnosis is made, prompt decisions must be made as to whether to give medical therapy alone, or that with percutaneous drainage or a nephrectomy. To evaluate the prognostic factors of cases with EPN, two classification systems has been reported by Wan et al[15] and Huang et al.[16] The former reported that patients with creatinine levels > 1.4 mg/dL and platelet counts $\leq$ 60,000/mm^3 were at high risk,[15] although later reported that patients with two or more risk factors (altered consciousness, thrombocytopenia, shock, acute renal failure) require aggressive treatment.[16]

Sodium-glucose transport proteins (SGLTs) have recently become a topic of significant interest in the field of type 2 diabetes. SGLT-2 inhibitors block reabsorption of filtered glucose, leading to glucosuria and improvement in glycemic control, although they are also associated with caloric loss, which has the potential to lead to weight loss.[17] In patients using an SGLT-2 inhibitor, glucose in the urine may encourage bacteria in the urinary tract to flourish, resulting in infection. Although several studies have shown increased incidence of UTI in patients taking an SGLT-2 inhibitor, others have reported that such treatment is accompanied by increased risk of genital infection, such as vulvovaginitis or balanitis, rather than UTI, which is related to induction of glucosuria.[18]

3. UTI with a neurogenic bladder

UTI in patients with a neurogenic bladder is characterized by clinical symptoms as well as laboratory findings of leukocyturia, bacteriuria, and positive urine culture. For diagnosis, a urine specimen should be obtained by a clean-catch midstream technique, via a newly inserted catheter, or bladder puncture. However, pyuria and bacteriuria have low specificity, because ASB is not rare in patients with a neurogenic bladder. Also, because of sensory and motor nerve paralysis, UTI in neurogenic bladder patients shows atypical symptomatology, including malaise and lethargy, new or increased urine leakage, increased spasticity, autonomic dysreflexia, loin pain, suprapubic pain, and dysuria.[19,20]

The spectrum of pathogens causing UTI in patients with a neurogenic bladder differs from that in patients with normal bladder function and is much broader. The majority of UTIs are generally caused by Gram-negative bacilli and enterococci, though sometimes by exogenous bacteria from the hospital environment and often by polymicrobial pathogens. Thus, urine culture and susceptibility testing should be performed before initiating antimicrobial therapy.[21] For selection of antimicrobial agents, regional differences in antibiotic resistance patterns must be taken into consideration.[22] The optimal duration of antimicrobial therapy ranges generally from 5 days to 14 days depending on severity, with 7 days most commonly used.[21,23]

For long-term management of neurogenic bladder dysfunction, the method used for bladder emptying is the most important issue. A lower rate of UTI and fewer complications are associated with intermittent catheterization as compared with indwelling urethral catheterization, supporting the notion that clean intermittent catheterization (CIC) is the best voiding method for reducing bacteriuria and urinary tract infections in patients with a neurogenic bladder.[24,25] Thus, CIC should be employed as a standard routine treatment for patients unable to empty their bladder. Furthermore, some reports have suggested that CIC using a hydrophilic-coated or prelubricated catheter may have additional advantages for reducing the risk of UTI in patients with spinal cord injury.[26,27]

Some studies have reported beneficial effects of antimicrobial prophylaxis in patients with a neurogenic bladder when given with a limited duration and under restricted conditions.[28] However, that therapy is generally not recommended, because its benefits are unproven and it has been associated with development of antimicrobial resistance.[21,22]

Bladder management in patients with neurogenic bladder, especially with spinal cord injury, includes not only bladder emptying by CIC but also effective pharmacologic therapy of neurogenic detrusor overactivity, using oral antimuscarinics.[29] Recently intradetrusor injections of botulinum toxin is applied in antimuscarinic refractory cases although this treatment has not been supported by a high level of evidence studies.[30] A spinal cord lesion also can cause concomitant neurological bowel dysfunction, resulting in constipation and fecal incontinence. Bowel management aimed at regular emptying of the bowels often relieves constipation and fecal soiling, and also reduces the risk of UTI.[31]

4. Catheter-associated UTI

Catheter-associated UTI (CAUTI) is generally defined as UTI associated with an indwelling urinary catheter; i.e., a drainage tube inserted into the urinary bladder through the urethra. A surveillance study conducted over a 6-year period by the International Nosocomial Infection Control Consortium of 422 intensive care unit (ICU) cases in 36 countries, 57% of which were in Asia, found that developing countries had a rate of 6.3 cases of CAUTI per 1000 urinary catheter-days, as compared with 3.3 per 1000 catheter-days in comparable ICU cases treated in the United States.[32] CAUTI in developing countries is also associated with a higher rate of antibiotic resistance shown by microbiological surveillance.

Biofilm that develops on a urinary catheter provides a favorable environment for bacterial proliferation and invasion. Bacteria can be introduced into the urinary tract via several routes, such as inoculation at the time of catheter insertion, an intraluminal ascent in the urinary catheter lumen, or an extraluminal route of ascent along the external surface of the urinary catheter.[33] Risk factors for CAUTI identified in prospective observational studies include duration of catheterization, female gender, anatomical or functional abnormalities of the urinary tract, insertion of the catheter outside the operating room, DM, poor catheter care, and breaks in the aseptic technique.[34–37] The most common uropathogen isolated is *Escherichia coli*, although other common enteric organisms have been isolated from patients who received short-term catheterization. *Proteus mirabilis* bacteriuria is often associated with catheter obstruction, although polymicrobial bacteriuria is commonly found in patients with long-term catheter use.[38]

The symptoms of CAUTI may include several signs, such as fever, hematuria, bladder irritability, suprapubic pain, and costovertebral angle tenderness indicating cystitis, as well as pyelonephritis, prostatitis, or epididymitis. However, distinction from catheter-associated ASB may be difficult in patients with a spinal cord

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injury who are unable to communicate due to illness, comorbidities, or extreme age.[39] Thus, a carefully performed physical examination including inspection and palpation of the perineal lesion, as well as a digital rectal examination are necessary. To exclude pneumonia or another febrile infectious disease at sites other than the urinary tract, image diagnostic tools such as ultrasound, x-ray, and CT are useful.

Significantly higher rates of antibiotic resistance have been found in Asia as compared with Europe and North America,[40] thus broad-spectrum antimicrobials should be empirically administered based on monitoring of local antibiotic resistance patterns by uropathogens. Such empiric antimicrobial therapy can be guided by recent prior urine culture results, when available.[41] A prospective randomized study showed that clinical and bacteriological outcomes were significantly improved when long-term indwelling catheters were replaced before initiation of antimicrobial therapy.[42] A few days after initiating that therapy, the effectiveness of the empirical drug should be assessed, with deescalation to the narrowest spectrum antimicrobial agent recommended, as guided by urine culture results.

Even though a variety of different types of urethral catheters are available, including those impregnated with silver or antibiotics, no randomized study has presented findings supporting a role for their use to prevent CAUTI related to long-term bladder drainage.[43] Alternative methods of bladder drainage with suprapubic catheter may be considered, but its evidence of efficacy in preventing CAUTI remains limited. Based on the clinical findings, suprapubic catheters are associated with a low incidence of urethral injury and stricture, but have similar rates of upper tract damage such vesicoureteral reflux, renal or bladder calculi, and symptomatic urinary tract infections as well as cancers, compared with urethral catheters.[44,45] Thus, to prevent CAUTI, it is strongly recommended to avoid unnecessary urinary catheterization and minimize the duration of catheterization by close surveillance.[46,47] To shorten the catheterization period, a nurse-generated or electronic-based reminder, as well as a stop-order system for removal of the urinary catheter have been reported to be effective.[48–50] Furthermore, urinary catheter care bundles and infection control programs have also been shown to reduce the rate of CAUTI, including educational and training regarding catheter indications, aseptic catheter insertion techniques, hygienic practices, and surveillance and performance feedback systems.[51,52] No recommendation can be given for routine use of antibiotic prophylaxis for prevention of CAUTI. Needless to say, routine use of antimicrobial prophylaxis for prevention of CAUTI for either short- or long-term urinary catheterization is not recommended, as that would result in an increase in resistant pathogens.[53–55]

4.1. Obstructive pyelonephritis secondary to stones

Obstructive pyelonephritis complicated by urolithiasis can be life-threatening, and requires prompt diagnosis and treatment, including medical as well as surgical intervention to rescue the affected patient. For prompt diagnosis of complicated UTI and accurate evaluation of physical condition, imaging diagnosis should be considered for males or any patient with a history of DM, urolithiasis, urological abnormality, relapsing UTI, febrile UTI, urine pH $\geq$7.0, and/or renal insufficiency.[56–58] Ultrasound, plain abdominal X-ray imaging, and abdominal and pelvic CT are the most commonly utilized imaging tools. A Japanese study of 1363 patients revealed that female patients outnumber males by 2.2 times, although the mortality rate for all affected patients was 2.3%. Multivariate analysis showed that the predictive risk factors for death from disease during hospitalization were age > 80 years, systemic inflammatory response syndrome (SIRS), disseminated intravascular coagulation status (DIC), disturbance of consciousness, and solitary kidney.[59]

E. coli is the most frequently isolated uropathogen, while others related include *P. mirabilis*, *Klebsiella*, and Enterococci, though some cases show multiple uropathogens. *Candida albicans* as well can be isolated from the urine of these patients.[59–61] Although a urine culture is definitely required prior to initiation of antimicrobial therapy, the results of a bladder urine culture do not always correlate with those of stone and renal pelvic cultures.[55] In febrile UTI with urosepsis cases, two blood culture examinations must be performed for differentiation between true bacteremia and contamination.

For decompression of the renal collecting system, retrograde ureteral catheterization (RUC) and percutaneous nephrostomy (PCN) are equally effective,[62] with the decision of which mode of drainage to choose primarily based on logistical factors such as surgeon preference and stone characteristics,[63] though patients who have undergone RUC may have a worse result as compared with those who have received PCN.[64] Some studies have shown that training background, favorable physician reimbursement, timing of intervention, and patient-specific factors are significant drivers in this decision-making process,[65,66] though neither modality has been demonstrated to be superior for promoting more rapid recovery after drainage.

5. Conclusion

A complicated UTI is a relapsing refractory condition, and can be life-threatening because of patient predisposing factors as well as the worldwide spread of multi-drug resistant bacteria. Attending physicians must choose wisely among appropriate treatment approaches based on a correct understanding of the etiologies, as well as available diagnostic strategies and treatment options for affected patients.

Conflicts of interest

The authors declare that they have no financial or nonfinancial conflicts of interest related to the subject matter or materials discussed in the manuscript.

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References

1. Nicolle LE. A practical guide to antimicrobial management of complicated urinary tract infection. *Drugs Aging* 2001;**18**:243–54.
2. Rubin RH, Shapiro ED, Andriole VT, Davis RJ, Stamm WE. Evaluation of new antiinfective drugs for the treatment of urinary tract infection. *Clin Infect Dis* 1992;**15**(Suppl. 1):S216–27.
3. Nicolle LE, Bradley S, Colgan R, Rice JC, Schaeffer A, Hooton TM. Infectious Diseases Society of America guidelines for the diagnosis and treatment of asymptomatic bacteriuria in adults. *Clin Infect Dis* 2005;**40**:643–54.
4. Zhanel GG, Harding GK, Nicolle LE. Asymptomatic bacteriuria in patients with diabetes mellitus. *Rev Infect Dis* 1991;**13**:150–4.
5. Harding GK, Zhanel GG, Nicolle LE, Cheang M, Manitoba Diabetes Urinary Tract Infection Study Group. Antimicrobial treatment in diabetic women with asymptomatic bacteriuria. *N Engl J Med* 2002;**347**:1576–83.
6. Brown JS, Wessells H, Chancellor MB, Howards SS, Stamm WE, Stapleton AE, et al. Urologic complications of diabetes. *Diabetes Care* 2005;**28**:177–85.
7. Muller LM, Gorter KJ, Hak E, Goudzwaard WL, Schellevis FG, Hoepelman AI, et al. Increased risk of common infections in patients with type 1 and type 2 diabetes mellitus. *Clin Infect Dis* 2005;**41**:281–8.

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8. Geerlings SE, Stolk RP, Camps MJ, Netten PM, Collet TJ, Hoepelman AI, et al. Risk factors for symptomatic urinary tract infection in women with diabetes. *Diabetes Care* 2000;**23**:1737–41.
9. Deli G, Bosnyak E, Pusch G, Komoly S, Feher G. Diabetic neuropathies: diagnosis and management. *Neuroendocrinology* 2013;**98**:267–80.
10. Pontin AR, Barnes RD. Current management of emphysematous pyelonephritis. *Nat Rev Urol* 2009;**6**:272–9.
11. Sentochnik DE, Eliopoulos GM. Infection in diabetes. In: Kahn R, Weir G, editors. *Joslins Diabetes mellitus*. 14th ed. Philadelphia: Lippincot Williams and Wilkins; 2005. p. 1022.
12. Grayson DE, Abbott RM, Levy AD, Sherman PM. Emphysematous infections of the abdomen and pelvis: a pictorial review. *Radiographics* 2002;**22**:543–61.
13. Aboumarzouk OM, Hughes O, Narahari K, Coulthard R, Kynaston H, Chlosta P, et al. Emphysematous pyelonephritis: time for a management plan with an evidence-based approach. *Arab J Urol* 2014;**12**:106–15.
14. Thomas AA, Lane BR, Thomas AZ, Remer EM, Campbell SC, Shoskes DA. Emphysematous cystitis: a review of 135 cases. *BJU Int* 2007;**100**:17–20.
15. Wan YL, Lo SK, Bullard MJ, Chang PL, Lee TY. Predictors of outcome in emphysematous pyelonephritis. *J Urol* 1998;**159**:369–73.
16. Huang JJ, Tseng CC. Emphysematous pyelonephritis: clinicoradiological classification, management, prognosis, and pathogenesis. *Arch Intern Med* 2000;**160**:797–805.
17. Bolinder J, Ljunggren Ö, Johansson L, Wilding J, Langkilde AM, Sjöström CD, et al. Dapagliflozin maintains glycaemic control while reducing weight and body fat mass over 2 years in patients with type 2 diabetes mellitus inadequately controlled on metformin. *Diabetes Obes Metab* 2014;**16**:159–69.
18. Johnsson KM, Ptaszynska A, Schmitz B, Sugg J, Parikh SJ, List JF. Vulvovaginitis and balanitis in patients with diabetes treated with dapagliflozin. *J Diabetes Complications* 2013;**27**:479–84.
19. Goetz LL, Cardenas DD, Kennelly M, Bonne Lee BS, Linsenmeyer T, Moser C, et al. International spinal cord injury urinary tract infection basic data set. *Spinal Cord* 2013;**51**:700–4.
20. Massa LM, Hoffman JM, Cardenas DD. Validity, accuracy, and predictive value of urinary tract infection signs and symptoms in individuals with spinal cord injury on intermittent catheterization. *J Spinal Cord Med* 2009;**32**:568–73.
21. Cardenas DD, Hooton TM. Urinary tract infection in persons with spinal cord injury. *Arch Phys Med Rehabil* 1995;**76**:272–80.
22. Morton SC, Vickrey BG. Antimicrobial prophylaxis for urinary tract infection in persons with spinal cord dysfunction. *Arch Phys Med Rehabil* 2002;**83**:129–38.
23. Lee MTG, Lee SH, Chang SS, Lee SH, Lee M, Fang CC, et al. Comparative effectiveness of different oral antibiotics regimens for treatment of urinary tract infection in outpatients: an analysis of national representative claims database. *Medicine* 2014;**93**:e304.
24. Dedeic-Liubovic A, Hukic M. Catheter-related urinary tract infection in patients suffering from spinal cord injuries. *Bosn J Basic Med Sci* 2009;**9**:2–9.
25. Ryu KH, Kim YB, Yang SO, Lee JK, Jung TY. Results of urine culture and antimicrobial sensitivity tests according to the voiding method over 10 years in patients with spinal cord injury. *Korean J Urol* 2011;**52**:345–9.
26. De Ridder DJ, Everaert K, Fernández LG, Valero JV, Durán AB, Abrisqueta ML, et al. Intermittent catheterisation with hydrophilic-coated catheters (SpeediCath) reduces the risk of clinical urinary tract infection in spinal cord injured patients: a prospective randomised parallel comparative trial. *Eur Urol* 2005;**48**:991–5.
27. Giannantoni A, Di Stasi SM, Scivoletto G, Virgili G, Dolci S, Porena M. Intermittent catheterization with a prelubricated catheter in spinal cord injured patients: a prospective randomized crossover study. *J Urol* 2001;**166**:130–3.
28. Salomon J, Denys P, Merle C, Chartier-Kastler E, Perronne C, Gaillard JL, et al. Prevention of urinary tract infection in spinal cord-injured patients: safety and efficacy of a weekly oral cyclic antibiotic (WOCA) programme with a 2 year follow-up—an observational prospective study. *J Antimicrob Chemother* 2006;**57**:784–8.
29. Sahai A, Cortes E, Seth J, Khan MS, Panicker J, Kelleher C, et al. Neurogenic detrusor overactivity in patients with spinal cord injury: evaluation and management. *Curr Urol Rep* 2011;**12**:404–12.
30. Hascoet J, Manunta A, Brochard C, Arnaud A, Damphousse M, Menard H, et al. Outcomes of intra-detrusor injections of botulinum toxin in patients with spina bifida: a systematic review. *Neurourol Urodyn* 2016. http://dx.doi.org/10.1002/nau.23025.
31. Ausili E, Focarelli B, Tabacco F, Murolo D, Sigismondi M, Gasbarrini A, et al. Transanal irrigation in myelomeningocele children: an alternative, safe and valid approach for neurogenic constipation. *Spinal Cord* 2010;**48**:560–5.
32. Wagenlehner FME, Cek M, Naber KG, Kiyota H, Bjerklund-Johansen TE. Epidemiology, treatment, and prevention of healthcare-associated urinary tract infections. *World J Urol* 2012;**30**:59–67.
33. Nicolle LE. Urinary catheter-associated infections. *Infect Dis Clin North Am* 2012;**26**:13–27.
34. Maki DG, Tambyah PA. Engineering out the risk for infection with urinary catheters. *Emerg Infect Dis* 2001;**7**:342–7.
35. Foxman B. The epidemiology of urinary tract infection. *Nat Rev Urol* 2010;**7**:653–60.
36. Platt R, Polk BF, Murdock B, Rosner B. Risk factors for nosocomial urinary tract infection. *Am J Epidemiol* 1986;**124**:977–85.
37. Barbadoro P, Labricciosa FM, Recanatini C, Gori G, Tirabassi F, Martini E, et al. Catheter-associated urinary tract infection: role of the setting of catheter insertion. *Am J Infect Control* 2015;**43**:707–10.
38. Warren JW. Catheter-associated urinary tract infections. *Int J Antimicrob Agents* 2001;**17**:299–303.
39. Tambyah PA, Maki DG. Catheter-associated urinary tract infection is rarely symptomatic: a prospective study of 1,497 catheterized patients. *Arch Intern Med* 2000;**160**:678–82.
40. Morrissey I, Hackel M, Badal R, Bouchillon S, Hawser S, Biedenbach D. A review of 10 years of the study for monitoring antimicrobial resistance trends (SMART) from 2002 to 2011. *Pharmaceuticals* 2013;**6**:1335–46.
41. MacFadden DR, Ridgway JP, Robicsek A, Elligsen M, Daneman N. The predictive utility of prior positive urine cultures. *Clin Infect Dis* 2014;**59**:1265–71.
42. Raz R, Schiller D, Nicolle LE. Chronic indwelling catheter replacement before antimicrobial therapy for symptomatic urinary tract infection. *J Urol* 2000;**164**:254–8.
43. Jahn P, Beutner K, Langer G. Types of indwelling urinary catheters for long-term bladder drainage in adults. *Cochrane Database Syst Rev* 2012;17;**10**:CD004997.
44. Hunter KF, Bharmal A, Moore KN. Long-term bladder drainage: suprapubic catheter versus other methods: a scoping review. *Neurourol Urodyn* 2013;**32**:944–51.
45. Katsumi HK, Kalisvaart JF, Ronningen LD, Hovey RM. Urethral versus suprapubic catheter: choosing the best bladder management for male spinal cord injury patients with indwelling catheters. *Spinal Cord* 2010;**48**:325–9.
46. Wald HL, Ma A, Bratzler DW, Kramer AM. Indwelling urinary catheter use in the postoperative period: analysis of the national surgical infection prevention project data. *Arch Surg* 2008;**143**:551–7.
47. West DA, Cummings JM, Longo WE, Virgo KS, Johnson FE, Parra RO. Role of chronic catheterization in the development of bladder cancer in patients with spinal cord injury. *Urology* 1999;**53**:292–7.
48. Meddings J, Rogers MAM, Macy M, Saint S. Systematic review and meta-analysis: reminder systems to reduce catheter-associated urinary tract infections and urinary catheter use in hospitalized patients. *Clin Infect Dis* 2010;**51**:550–60.
49. Fakih MG, Dueweke C, Meisner S, Berriel-Cass D, Savoy-Moore R, Brach N, et al. Effect of nurse-led multidisciplinary rounds on reducing the unnecessary use of urinary catheterization in hospitalized patients. *Infect Control Hosp Epidemiol* 2008;**29**:815–9.
50. Cornia PB, Amory JK, Fraser S, Saint S, Lipsky BA. Computer-based order entry decreases duration of indwelling urinary catheterization in hospitalized patients. *Am J Med* 2003;**114**:404–7.
51. Lo E, Nicolle LE, Coffin SE, Gould C, Maragakis LL, Meddings J, et al. Strategies to prevent catheter-associated urinary tract infections in acute care hospitals: 2014 update. *Infect Control Hosp Epidemiol* 2014;**35**:464–79.
52. Saint S, Greene MT, Kowalski CP, Watson SR, Hofer TP, Krein SL. Preventing catheter-associated urinary tract infection in the United States: a national comparative study. *JAMA Intern Med* 2013;**173**:874–9.
53. Lusardi G, Lipp A, Shaw C. Antibiotic prophylaxis for short-term catheter bladder drainage in adults. *Cochrane Database Syst Rev* 2013;**7**:CD005428.
54. Marschall J, Carpenter CR, Fowler S, Trautner BW. Antibiotic prophylaxis for urinary tract infections after removal of urinary catheter: meta-analysis. *BMJ* 2013 Jun 11;**346**:f3147. http://dx.doi.org/10.1136/bmj.f3147.
55. Niël-Weise BS, van den Broek PJ, da Silva EMK, Silva LA. Urinary catheter policies for long-term bladder drainage. *Cochrane Database Syst Rev* 2012;**8**:CD004201.
56. Demertzis J, Menias CO. State of the art: imaging of renal infections. *Emerg Radiol* 2007;**14**:13–22.
57. van Nieuwkoop C, Hoppe BP, Bonten TN, Van't Wout JW, Aarts NJ, Mertens BJ, et al. Predicting the need for radiologic imaging in adults with febrile urinary tract infection. *Clin Infect Dis* 2010;**51**:1266–72.
58. Sørensen SM, Schønheyder HC, Nielsen H. The role of imaging of the urinary tract in patients with urosepsis. *Int J Infect Dis* 2013;**17**:e299–303.
59. Hamasuna R, Takahashi S, Nagae H, Kubo T, Yamamoto S, Arakawa S, et al. Obstructive pyelonephritis as a result of urolithiasis in Japan: diagnosis, treatment and prognosis. *Int J Urol* 2015;**22**:294–300.
60. Pearle MS, Pierce HL, Miller GL, Summa JA, Mutz JM, Petty BA, et al. Optimal method of urgent decompression of the collecting system for obstruction and infection due to ureteral calculi. *J Urol* 1998;**160**:1260–4.
61. Yoshimura K, Utsunomiya N, Ichioka K, Ueda N, Matsui Y, Terai A. Emergency drainage for urosepsis associated with upper urinary tract calculi. *J Urol* 2005;**173**:458–62.
62. Mariappan P, Loong CW. Midstream urine culture and sensitivity test is a poor predictor of infected urine proximal to the obstructing ureteral stone or infected stones: a prospective clinical study. *J Urol* 2004;**171**:2142–5.
63. Preminger GM, Tiselius HG, Assimos DG, Alken P, Buck C, Gallucci M, et al., EAU/AUA Nephrolithiasis Guideline Panel. 2007 guideline for the management of ureteral calculi. *J Urol* 2007;**178**:2418–34.
64. Mokhmalji H, Braun PM, Martinez Portillo FJ, Siegsmund M, Alken P, Köhrmann KU. Percutaneous nephrostomy versus ureteral stents for diversion of hydronephrosis caused by stones: a prospective, randomized clinical trial. *J Urol* 2001;**165**:1088–92.
65. Matlaga BR. How do we manage infected, obstructed hydronephrosis? *Eur Urol* 2013;**64**:93–4.
66. Childs MA, Rangel LJ, Lingeman JE, Krambeck AE. Factors influencing urologist treatment preference in surgical management of stone disease. *Urology* 2012 May;**79**(5):996–1003. http://dx.doi.org/10.1016/j.urology.2011.11.024 [Epub 2012 Jan 13].

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